

Neurobiology and the Law: A Role in Juvenile Justice?

Staci A. Gruber & Deborah A. Yurgelun-Todd*

Human behavior is determined by a complex interaction between biology and experience. In childhood, it is clear that specific biological milestones need to be reached for key behaviors to emerge. As we move into adolescence, it is more difficult to recognize the relationship between biological underpinnings and behavior. Just how old do you have to be to make a good decision? Determining the point at which someone is able to fully understand the consequences of his actions and be held accountable for such is critical to making and enforcing laws. A closer look at the neurobiology of adolescence and the processes involved in brain development underscore the importance of considering a number of factors when evaluating whether juveniles may be "held accountable" for all of their actions. While parental guidance, education and peer values undoubtedly play important roles in adolescent behavior, the integrity of the brain, particularly the prefrontal cortical region is of special importance. Data from recent investigations provide evidence that brain maturation continues well past where we once thought adolescence ends. Accordingly, the developmental factors which influence decision-making in adolescents may result in choices which are suggestive of cortical immaturity, poor judgment and impulsivity. It is reasonable then, to assume that all significant factors, including chronological age, nature and severity of the crime, previous history, and neurobiologic stage of development should be considered when dealing with juvenile offenders.

I. INTRODUCTION

Human behavior is determined by a complex interaction between biology and experience. In childhood, it is clear that specific biological milestones need to be reached for key behaviors to emerge. For example, walking requires muscle development, neural coordination and practice. As we move into adolescence it is more difficult to recognize the relationship between biological underpinnings and the increased functionality associated with mature behavior. The concept of childhood and adolescence is predominantly a cultural and social phenomenon, and, as a result, clear beginning and end points are not easily referenced by physical milestones. The exact definition of a child varies, but it is often referenced as "someone who is a young human" or a person who is between birth

* Dr. Gruber is the Associate Director and Dr. Yurgelun-Todd is the Director of the Cognitive Neuroimaging and Neuropsychology Laboratory of McLean Hospital, Harvard Medical School. Comments or questions may be directed to Dr. Gruber at gruber@mclean.harvard.edu.

and puberty. Accordingly, adolescence is often defined as a transitional stage of development between childhood and full adulthood, representing the period of time during which a person may physically be considered an adult but may not in fact be emotionally at full maturity. Of course, the definition of maturity is also culturally defined, and not based on any specific neurobiologic evidence. The ages that are considered to be part of adolescence vary by culture; even within the United States there is dispute regarding absolute age ranges. However, organizations like the Center for Disease Control (CDC) define adolescence as anyone between the ages of ten and twenty-four years of age (Virginia Department of Health—Office of Family Health Services, 2005).

Just how old do you have to be to make a good decision? Unfortunately, despite an increasing amount of research on the development of the human brain, the answer is still unclear. Since so many of our cultural and sociological practices, like getting a driver's license, buying alcohol, enlisting in the military, voting in elections, getting a marriage license, or even getting into a movie are based on chronological age, determining when an individual might arrive at the "age of reason" is critically important. Further, determining the point at which someone is able to fully understand the consequences of and be held accountable for his actions is critical to making and enforcing laws. A closer look at the neurobiology of adolescence and the processes involved in brain development underscore the importance of considering a number of factors when evaluating whether juveniles may be held accountable for all of their actions. While parental guidance, education, and peer values undoubtedly play important roles in adolescent behavior, the integrity of the brain, particularly the prefrontal cortical region is of special importance. The prefrontal cortex in the portion of the brain located behind the eyebrows is the forward most portion of the brain. Divided into the dorsolateral, orbitofrontal and mesial prefrontal areas, this brain region has been implicated in planning complex cognitive behaviors, personality expression and moderating correct social behavior.

II. THE PREFRONTAL CORTEX

The cerebral hemispheres of primates can be divided into a frontal and posterior portion at the level of the central sulcus. The posterior portion subserves perception, sensation, and perceptual memory functions and the frontal portion governs action and motor memory functions. It appears that increasingly complex actions or functions are organized in a hierarchical manner within the frontal lobe, and that the most complex and novel action domains depend upon the integrity of the prefrontal cortex (PFC). The PFC has a pivotal role in the development and execution of novel thoughts and behaviors which are thought to be represented by neural networks as "abstract schema" or mental representations. For example the mental representations of "insight," "judgment," "winning," and "goals" are all supported by this brain region. The simpler action components of these schema are felt to be represented by neural networks in frontal and subcortical regions

which constitute lower levels of the motor hierarchy. During execution of an action plan, the flow of neural activity is generally from the prefrontal to the premotor and ultimately the motor cortex. However, these areas are interconnected in a reciprocal manner, both with each other and other deeper brain areas. In this way, both serial (sequential or linear) and parallel (simultaneous processing by multiple regions) processing occur at the same time.

The frontal cortex has been shown to play a major role in the performance of executive functions including short term or working memory, motor set and planning, attention, inhibitory control and decision making (Lezak, 2004; Goldberg, 2001; Luria, 1966). These functions are subserved by reciprocal connections between the prefrontal cortex and posterior cortical regions as well as subcortical regions. Working memory involves the capacity to keep new sensory or motor information, or a newly activated memory "on line" for a short period of time so that the information present can be processed and acted upon. This process is mediated primarily by the dorsolateral prefrontal region. The capacity to maintain information "on line" is thought to result from the repetitive activation of specific neuronal networks via reverberating circuits or connections with shared access to the information.

Attention or motor set and planning are mediated by the medial prefrontal cortex. These functions require the selection of a particular motor act to be performed from an existing repertoire of motor acts in motor memory and the preparation of various related sensory and motor systems for the performance of this act. Inhibition is mediated primarily by the orbitomedial prefrontal cortex and acts to suppress extraneous and distracting motor or sensory stimuli or memories that might interfere with performance of the task at hand. The process of decision-making requires inhibitory function as well as attention and planning, and is mediated by a complex interaction of the frontal systems described. Each of these functions, most notably the ability to make decisions, has an important impact on juvenile behavior. For example, the choice of whether or not to engage in illegal or risky behavior requires an individual to have some facility and cognitive appreciation of the functions described above. Of particular importance is the fact that each of these functions draws upon diffuse neural networks linking multiple cortical and subcortical areas which must reach an appropriate maturational level for an individual to utilize good judgment and make good decisions.

Previous studies have shown that the capacity to perform well on simple tests that utilize both working memory and inhibitory capacity progresses steadily in infant monkeys throughout the first four months of life, and in human infants from the seventh to the twelfth month of life (Piaget, 1954; Jacobsen, 1935; Diamond & Goldman-Rakic, 1989; Diamond, 1996). This is the age range at which object constancy develops; it has been suggested that object constancy is simply one facet of working memory capacity in the infant (Diamond, 1991). This term refers to the fact that when children develop object constancy, they look for objects in the last place it was seen, instead of its original position. Adolescent or adult monkeys with lesions of the dorsolateral prefrontal cortex do not exhibit object constancy,

but monkeys with lesions of the parietal cortex or hippocampus do *not* have difficulty with these tasks (Diamond & Goldman-Rakic, 1989). Although great maturational strides occur in the prefrontal cortex of human infants during the first year of life, there is now considerable evidence that the prefrontal cortex is not fully mature until much later. In fact, in an investigation by Sowell and colleagues utilized structural magnetic resonance imaging techniques (MRI) to examine brain maturation in a group of adolescents (twelve to sixteen years old) and young adults (twenty-three to thirty years old), the authors report both a temporal and spatial progression of post adolescent maturation into the frontal lobes, underscoring that frontal lobe development continues into at least the third decade of life (Sowell et al., 1999).

In humans, the *basic* cytoarchitecture, or arrangement of cells in the tissue of the prefrontal cortex, is in place by the seventh intrauterine month and essentially complete by birth (Conel, 1939; Larroche & Amiel, 1966; Mrzljak et al., 1990). While the basic cellular structure may be in place for the prefrontal cortex early in life, maturation of neurons and the establishment and refinement of cell contacts continues for many years. For example, the differentiation of cell types within the hippocampus believed to mediate associative memory processes continues until puberty (Mrzljak et al., 1990). This example demonstrates that key anatomical aspects of the memory process are not developed until puberty. Furthermore, the PFC and other association areas are the *last* regions to begin myelination during the perinatal stage and the *last* regions to complete it (Yakovlev & Lecours, 1967; Sowell et al., 1999). Therefore, while the basic cellular structure may be in place for the prefrontal cortex early in life, the connectivity and efficiency of these connections has been shown to continue developing throughout adolescence and early adulthood (Huttenlocher & Dabholkar, 1997).

Given the neurobiologic findings described above, it is not surprising that adolescence is a critical period for brain development, characterized by significant decreases in cortical gray matter and increases in white matter (Giedd et al., 1996; Giedd et al., 1999; Jernigan et al., 1991; Pfefferbaum et al., 1994; Yurgelun-Todd et al., 2002). White matter is distinguished in that it is composed of nerve fibers often covered with myelin. This is as opposed to gray matter, which is composed primarily of nerve cell bodies. Generally, white matter can be understood as the parts of the brain responsible for information transmission, whereas gray matter is responsible for information processing. Giedd and colleagues reported that the increase in white matter occurs linearly during development, whereas gray matter increases during preadolescence, peaking in the frontal cortex around age twelve, but then decreases during post adolescence (Giedd et al., 1999). Likewise, Sowell and colleagues reported that the largest maturational changes observed between twelve to sixteen and twenty-three to thirty years occurred in dorsal, medial, and lateral regions of the frontal lobes, as compared to parietal and occipital lobes (Sowell et al., 1999). Indeed, it has been well established that reductions in gray matter presumably reflect, in part, increased myelination, which may be associated with age-related improvements in cognitive processing (Yurgelun-Todd et al.,

2002). Diffusion Tensor Imaging (DTI) is a non-invasive technique that is used to characterize structural properties of matter or tissues from measurements of water diffusion. A recent DTI study has demonstrated that anisotropy, a measure reflecting myelin-related restriction of water diffusion across axons (threadlike process of a neuron), in frontal white matter was significantly lower in children than in adults, suggesting less myelination in children (Klingberg et al., 1999).

While it is clear that development is associated with progressive increases in the ratio of cerebral white-to-gray-matter volume, the precise ways in which these changes relate to cognitive development has been a critical area of investigation. Given previous evidence that gray matter tends to decline during adolescence, while white matter continues to increase well into adulthood (Pfefferbaum et al., 1994), Yurgelun-Todd and colleagues examined whether greater volume of white matter would be associated with better performances on a battery of standard neurocognitive tests (Yurgelun-Todd et al., 2002; see Table 1). The authors examined the relationship between cerebral tissue volume and cognitive performance in healthy adolescents using morphometric magnetic resonance imaging (MRI), and found that the proportional volumes of white matter, gray matter, and cerebrospinal fluid were significantly associated with variability in cognitive performances on several cognitive factors. Overall, greater volume of white matter and concomitantly reduced gray matter volume was associated with more efficient and rapid processing of information and generally stronger verbal skills in our sample of adolescents. The significant correlations between white matter volume and processing speed are consistent with evidence suggesting that increased myelination of axons produces faster conduction velocity of neural signals (Waxman & Foster, 1980) and more efficient processing of information (Bartres-Faz et al., 2001), and further suggest that some of the increased cognitive abilities characteristic of adult maturation may be associated with developmental increases in relative white matter volume.

Table 1. Correlations Between Regional Tissue Volume and Cognitive Factors

Tissue Volume ¹	Cognitive Factor			
	Age	Processing Speed/Efficiency	Verbal Ability	Mental Flexibility Working Memory
Total Sample (<i>n</i> = 30)				
Age	--	.20	.56**	.19
Gray Matter	-.11	-.50**	-.32	.12
White Matter	.14	.52**	.36*	-.09
Cerebrospinal fluid	.36*	-.15	-.42*	-.30
Total Brain Volume	.16	.25	.19	.10
Gray Matter Asymmetry	-.07	-.38*	-.03	-.24
White Matter Asymmetry	.05	.05	.07	.08
Males (<i>n</i> = 10)				
Age	--	.40	.61	.23
Gray Matter	-.40	-.96**	-.58	.18
White Matter	.39	.96**	.63*	-.16
Cerebrospinal fluid	-.60	-.25	-.89**	-.30
Total Brain Volume	.42	.71*	.39	.19
Gray Matter Asymmetry	.33	-.73*	.42	-.25
White Matter Asymmetry	-.68	-.05	.08	-.13
Females (<i>n</i> = 20)				
Age	--	.11	.56**	.19
Gray Matter	.15	.07	.05	.05
White Matter	-.13	-.06	-.05	.00
Cerebrospinal fluid	-.22	-.11	-.08	-.32
Total Brain Volume	.01	.08	.13	.09
Gray Matter Asymmetry	-.11	-.31	-.08	-.36
White Matter Asymmetry	.17	.05	.03	.21

Note.¹ Tissue volumes are corrected for total intracranial volume, **p* < .05, ***p* < .01

Findings from Yurgelun-Todd and others suggest that these tissue volume changes are associated with measurable performances on cognitive tasks, and that reduced cerebral tissue volume, as evidenced by greater proportions of cerebrospinal fluid, is also associated with poorer cognitive performance, particularly for tasks measuring verbal abilities (Yurgelun-Todd et al., 2003; Yurgelun-Todd et al., 2002). Further, it appears that changes in white matter volume and concomitant decreased gray matter volume during adolescence are associated with stronger performances on cognitive tasks assessing the speed of information processing and general verbal abilities. These findings complement

other recent neurobiologic studies suggesting that the development of cognitive abilities appears to be related to structural and physiologic brain changes that occur during childhood and adolescence (Gomez-Perez et al., 2003; Sowell et al., 2001; Yurgelun-Todd et al., 2003; Yurgelun-Todd et al., 2002).

While structural studies have helped document brain changes occurring during adolescence, the relationship between structural brain change and resultant behavior has not always been clear. Recently, there has been an increase in the awareness of impulsive and often dangerous behavior in juveniles, a fact which has underscored the need for understanding the healthy development of emotional processing in adolescents. In contrast to the considerable literature on childhood and adolescent emotional development, relatively limited research exists on the neurobiological changes that occur during the transition between childhood and early adulthood. Fortunately, many of the developmental questions that were previously impossible to answer through traditional psychological studies can now be addressed through the use of newly developed brain imaging technology such as functional magnetic resonance imaging (fMRI). While a number of imaging techniques based on methods using x-rays or other ionizing radiation have been developed and used for studying the brain in subjects with neurological disorders, these methods are not well suited for children and adolescents. In contrast, magnetic resonance scanning is non-invasive and is free of ionizing radiation, allowing subjects to complete multiple experiments or several repetitions of the same experiment without risk. With this technology, it is now possible to obtain detailed brain images that reveal the specific areas and circuits within the brain which are involved in mental processes (Yurgelun-Todd & Renshaw, 2000).

A number of fMRI studies have examined whether the functional neuroanatomy underlying executive processing differs between children and adults; however, results of studies examining age-related differences in prefrontal brain activation have been inconsistent. Several investigations have reported similar patterns of prefrontal brain activity in children and adults on tasks of working memory (Nelson et al., 2000; Casey et al., 1995), response inhibition (Casey et al., 1997; Luna et al., 2001), and verbal fluency (Gaillard et al., 2000). A number of studies have underscored differences between these age groups on similar tasks of executive function, with children failing to utilize the same prefrontal brain areas as adult subjects (Thomas et al., 1999; Rubia et al., 2000; Bunge et al., 2002; Schlaggar et al., 2002). A number of methodological differences between studies may have contributed to these conflicting findings, including task performance mismatch between age groups and the absence of an adult comparison group in some investigations (Casey et al., 1995; Nelson et al., 2000). Notably, the only study that segregated performance from age-related prefrontal brain activation, (Schlaggar et al., 2002), found that adults but not children significantly activated the dorsal prefrontal cortex on a single-word processing task. While other age-group differences in prefrontal activation were also found, they were related to *accuracy* of task performance independent of age. The results of this study underline the importance of separating age-group

differences in brain activation into those attributable to maturation versus those secondary to accuracy of performance (Schlaggar et al., 2002).

Studies that have quantified the magnitude of prefrontal activation during tests of executive function have reported linear increases from childhood through young adulthood in the superior (Klingberg et al., 1999), middle (Rubia et al., 2000; Adleman et al., 2002), and inferior (Rubia et al., 2000) portions of prefrontal cortex. Klingberg and colleagues reported that the positive correlation between increasing age and magnitude of activation persists after controlling for performance accuracy (Klingberg et al., 1999). In contrast, one study reports a trend in the opposite direction with children tending to activate the right inferior frontal gyrus, an area located in the inferior portion of the frontal lobe (just behind the bridge of the nose), more powerfully than adults during word generation (Gaillard et al., 2000). Furthermore, an event-related fMRI study characterized age-differences in brain activation between children (ages eight to twelve) and adults on measures of cognitive control and showed that children were more susceptible to interference and less able to inhibit inappropriate responses than adults (Bunge et al., 2002). In addition, children exhibited immature prefrontal activation depending on the type of cognitive control required. Taken together, these studies suggest that cerebral maturation may be related to improved cognitive functioning. Although, little is known regarding the changes in cognitive functioning that occur prior to the onset of puberty versus those that occur within the few years following puberty, but prior to adulthood.

In a study designed to evaluate maturational changes associated with emotional response, Killgore and colleagues utilized fMRI techniques to test the hypothesis that adolescent development is associated with increased modulation of limbic system responsiveness by prefrontal inhibitory mechanisms (Hariri et al., 2000; Rubia et al., 2000; Killgore et al., 2001). Adolescent and adult subjects were presented with a fearful face perception task. The results demonstrated that in adult subjects, a significant increase in dorsolateral prefrontal cortex (DLPFC) activation during the viewing of fearful faces was detected, whereas the adolescent subjects showed no increase in prefrontal activation during the task. Of particular interest, we found that the adults showed lower activation within the amygdala relative to the adolescents, suggesting that adult maturation of the DLPFC was associated with reduced amygdala activity. Furthermore, within the adolescent sample, chronological age was significantly correlated with greater normalized signal intensity within the DLPFC ($r = .58$, $p = .02$), suggesting that prefrontal activity increases with adolescent maturation.

In a more recent investigation, the finding of increased frontal activation during conscious affective face processing has been replicated in a new group of subjects using a new data analytic approach (see Figures 1 and 2). Using a software package that reconstructs brain imaging data from each subject into a group map in 3-dimensional space, brain activation can be examined in association with age-related changes. During the perception of fearful facial affect, a significant positive relationship between age and right dorsolateral prefrontal

cortical activity is noted (see Figure 1). During the perception of happy facial affect, a significant positive relationship between age and activation within the anterior cingulate, an area of the brain tucked into the crease between the two hemispheres of the brain, is demonstrated (see Figure 2). The anterior cingulate has been linked to multiple processes, including decision making, evaluation of outcome, and inhibition. Results from this study suggest that during adolescent development, the amount of “work” carried out by frontal regions increases with age (Killgore & Yurgelun-Todd, 2005).

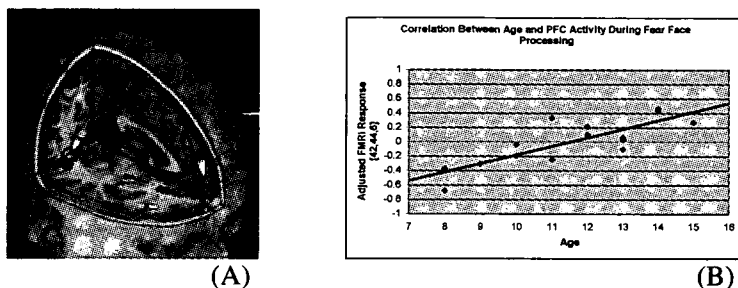


Figure 1. (A) Illustrates increased activation in the right dorsolateral prefrontal cortex during the perception of fearful affect. (B) Scatterplot demonstrates the significant age-correlated activation seen in the right dorsolateral prefrontal cortex during the perception of fearful affect.

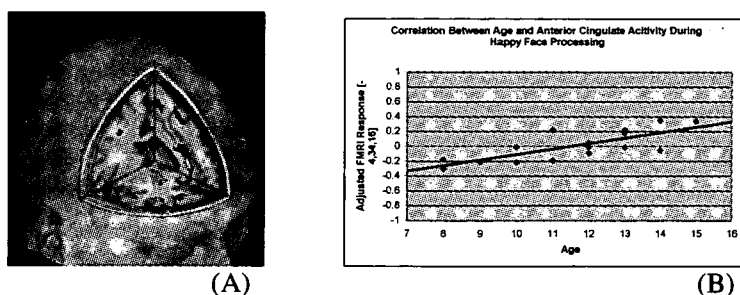


Figure 2. (A) Increased activation in the anterior cingulate during the perception of happy affect. (B) Scatterplot demonstrates the significant age-correlated activation in the anterior cingulate cortex during the perception of happy affect.

Difficulty with executive cognitive functioning and behavioral self-regulation, including difficulties with planning, attention, foresight, abstract reasoning, judgment, self-monitoring, and motor control, have been found to be present in adolescents. This is of particular importance, as neurobiological studies, including research measuring cerebral metabolic changes and rates of glucose utilization during cortical development, indicate that the cerebral cortex undergoes a dynamic course of metabolic maturation that persists at least until the age of eighteen

(Chugani, 1998). Moreover, studies comparing adult and adolescent cortical function indicate that adolescents process information differently, often enlisting different brain regions than do adults (Baird et al., 1999; Van der Stelt et al., 1998; Meyer-Lindenberg, 1996; Killgore et al., 2001; Killgore & Yurgelun-Todd, 2005). An adolescent's level of cortical development may therefore be directly related to her or his ability to perform well in situations requiring executive cognitive skills. Younger, less cortically mature adolescents may be more at risk for engaging in impulsive behavior than their older peers for two reasons. First, their developing brains are more susceptible to the neurological effects of external influences such as peer pressure. Second, they may make poor decisions because they are cognitively less able to select behavioral strategies associated with self-regulation, judgment, and planning that would reduce the effects of environmental risk factors for engaging in such behaviors.

III. JUVENILES AND DECISION-MAKING

The process of decision-making is surprisingly complex as it relies heavily on an interconnected neural system. In fact, individuals must be able to complete multiple processes for even the most seemingly simple decisions. This includes the perception of the stimuli as well as the situation, "holding" the set of response options online, assessing the implication of each option, and finally, the selection of the best option for the given situation (Braver & Bongiolatti, 2002). The higher order or executive components which are involved in this process include selective attention and short-term storage of information, inhibition of response to irrelevant information, initiation of response to relevant information, self-monitoring of performance, and changing internal and external contingencies in order to move towards the ultimate goal. These executive functions have been attributed to functions mediated by the frontal cortex (Daffner et al., 2000; Miller et al., 2000; Gruber et al., 2002; Gruber et al., 2004). Anomalous functioning or incomplete development of the prefrontal cortex has been documented to impair an individual's ability to monitor and inhibit behavior, and make effective decisions, and may therefore lead to inappropriate, impulsive behavior (Band et al., 2000; Killgore & Yurgelun-Todd, 2005; Rubia et al., 2000). Further, the documentable differences in processing affective and cognitive stimuli reported between adolescents and adults underscores the likelihood that both social and emotional influences, as well as processing abilities, affect juvenile behavior and their ability to make decisions. It follows, therefore, that if a juvenile's frontal cortex is not fully mature, he or she may make bad decisions reflective of an inability to adequately consider options and appreciate consequences.

The juvenile justice system was initially designed to reform American policies regarding youthful offenders. Early changes to the justice system were made under a conviction that society had a responsibility to recover the lives of its young offenders before they became absorbed in criminal activity (Schetky & Benedek, 2002). The juvenile justice system initially exercised its authority within

a *parens patriae* role. The current system, which now separates juvenile and adult criminal offenders, is based on two premises. First, adolescents as a group are less able to make good decisions based on mature levels of judgment, and as a result are less responsible for their actions, and second, are more likely to be receptive and responsive to treatment, thereby rendering them more likely to benefit from rehabilitation (Scott & Grisso, 1997). Despite the recognition that adolescents may differ from adults in their capacity to make good decisions and be successfully rehabilitated, many states have *lowered* the age limit for criminal prosecution with some having no lower age limit at all (Griffin, 2003). Nevertheless, the preponderance of the biological evidence indicates that profiles of adolescent functioning differ from those of adults.

Consider our current legal system. If a patient with bipolar disorder in the midst of a manic episode was arrested for driving erratically, narrowly missing pedestrians, and shouting insults out of the window of his car, the first point a defense attorney would make to a judge is that the client has a neurobiologic condition that renders him unable to modulate his behavior appropriately. A discussion of treatment, past and present might follow, as would some consideration of the client's behavioral history, and potential contributory factors to the incident. While the patient's behavior may not be deemed socially acceptable, it is difficult to imagine that some consideration of the underlying disorder would not be raised. Neuroscientific methods enable us to examine specific behavior or conditions in a way that was previously impossible. It is now understood that many psychiatric disorders, once thought or assumed to be the result of environmental or social factors, are in fact the result of differences in brain structure or function. Consider the same behavior exhibited by the bipolar patient being attributed to a seemingly healthy sixteen-year-old male. It is likely that an important consideration, namely the neurobiologic circumstances that have impacted the episode, has been forgotten. After all, this is a healthy adolescent boy, ostensibly free from any biologically based condition.

Based on neurobiological data alone, it is clear that children and adolescents are different both structurally and functionally from adults. In addition to documentable alterations which change during the trajectory of normal development, data from recent investigations provide evidence that brain maturation continues well past adolescence. Accordingly, the developmental factors which influence decision-making in adolescents may result in choices which are suggestive of cortical immaturity, poor judgment, and impulsivity. Is it fair then, for our society to consider adolescent offenders in the same way as adult offenders, or are they somehow less responsible given the ongoing "condition" of development?

The United States Supreme Court recently held in *Roper v. Simmons*, that it was unconstitutional to execute an offender for a crime committed when he was under the age of eighteen, primarily because of evidence from neurobiologic and neuropsychological investigations demonstrating the developmental immaturity of these offenders as a group. This ruling underscores the importance of viewing

adolescent offenders as fundamentally different from adult offenders. Further, given the variability inherent to the developmental process, the ruling also lends support to the consideration of additional mitigating circumstances. Factors including cognitive function, psychiatric status, and drug and alcohol use have all been linked to the ability to make decisions, even in healthy adult subjects. These areas must therefore be considered when evaluating the juvenile offender.

What advice can we offer attorneys and policy makers about children and adolescents who commit criminal acts, especially those marked by “impulsivity” or an unwillingness or inability to actively inhibit inappropriate behavior? Careful consideration of individual circumstances, including psychiatric status, substance use/abuse history, physical and emotional trauma, genetic predisposition for psychiatric or developmental disorders, and other factors must be clearly documented and entered into the client’s case file. A defense attorney representing a juvenile accused of committing a crime might take the following steps to help elucidate the state of mind and “baseline” levels of functioning in his client:

1. Obtain a comprehensive neuropsychological evaluation, which should include both objective and projective (which requires subjective interpretation and takes into consideration psychological intent and motivation) testing. One example of objective psychological testing is an examiner asking a subject to repeat a series of numbers in a particular order; an example of a projective test question would be an examiner asking the subject what a particular image or story makes them think of. These results should be summarized in a report. Individual test results may then be examined in relation to school records, with a special focus on potential areas of dysfunction which are longstanding, and areas of behavior which appear to have deteriorated over time.
2. Obtain all medical, psychological or psychiatric and academic records. Review the records for evidence of previous physical or emotional injury/trauma. Document evidence of difficulty in school which may inform the current situation. For example, if a juvenile offender has a history of poor school performance and poor attention, this may affect his/her ability to attend to current “risky” situations and evaluate circumstances.
3. If indicated on the basis of neuropsychological and medical evaluations, request a clinical MRI scan to evaluate overall brain integrity at the time of the incident. This scan should be read and interpreted by a clinical neuroradiologist to determine whether any evidence for organicity (i.e., organic brain syndrome, major structural abnormality), generalized atrophy or any other abnormality is present.

Chronological age does not dictate an individual’s level of social, emotional, or even physical maturity. Each of us has encountered young adults who are “mature” and seem much older than their stated age, and adults in their late thirties

or early forties who still act as if they are twelve. Since there is no clear method for determining "maturity" or "emotional adulthood," in our society, we are forced to consider other markers rather than simple chronological age. Recent neurobiologic investigations have begun to clarify some of the reasons why adolescents are not able to plan carefully, utilize good judgment, and practice behavioral inhibition when faced with difficult situations that often require a near immediate decision. Multiple factors, including neurobiology, social, economic, and psychological influences, all contribute to the complicated issue of juvenile behavior and culpability. Regardless of whether an individual supports the death penalty, it is reasonable to assume that all significant factors, including chronological age, nature and severity of the crime, previous history, and neurobiologic stage of development should be considered when dealing with juvenile offenders. Studies of adolescents which examine the relationship between structural and functional brain changes and the ability to make decisions are needed so that we may more carefully document maturational changes.

References

- ADLEMAN, N. E., MENON, V., BLASEY, C. M., WHITE, C. D., WARSOFSKY, I. S., GLOVER, G. H., & REISS, A. L. (2002). A Developmental fMRI Study of the Stroop Color-Word Task. *NeuroImage*, 16(1), 61–75.
- AKERT, K. (1964). Comparative anatomy of frontal cortex and thalamofrontal connections. In J. M. Warren & K. Akert (Eds), *The frontal granular cortex and behavior*. New York: McGraw Hill, 372–396.
- BAIRD, A. A., GRUBER, S. A., FEIN, D. A., MAAS, L. C., STEINGARD, R. J., RENSHAW, P. F., COHEN, B. M., & YURGELUN-TODD, D. A. (1999). Functional Magnetic Resonance Imaging of Facial Affect Recognition in Children and Adolescents. *J Am Acad Child Adolesc Psychiatry*, 38(2), 195–199.
- BAND, G. P., VAN DER MOLEN, M. W., OVERTOOM, C. C., & VERBATEN, M. N. (2000). The Ability to Activate and Inhibit Speeded Responses: Separate Developmental Trends. *J Exp Child Psychol*, 75(4), 263–290.
- BARTRES-FAZ, D., CLEMENTE, I. C., & JUNQUE, C. (2001). White matter changes and cognitive performance in aging. *Revista de Neurologia*, 33(4), 347–353.
- BARTZOKIS, G., BECKSON, M., LU, P. H., NUECHTERLEIN, K. H., EDWARDS, N., & MINTZ, J. (2001). Age-related changes in frontal and temporal lobe volumes in men: a magnetic resonance imaging study. *Arch Gen Psychiatry*, 58(5), 461–465.
- BRAVER, T. S., & BONGIOLATTI, S. R. (2002). The Role of Frontopolar Cortex in Subgoal Processing During Working Memory. *NeuroImage*, 15(3), 523–536.
- BUNGE, S. A., DUDUKOVIC, N. M., THOMASON, M. E., VAIDYA, C. J., & GABRIELI, J. D. (2002). Immature Frontal Lobe Contributions to Cognitive Control in Children: Evidence from fMRI. *Neuron*, 33(2), 301–311.
- CASEY, B. J., CASTELLANOS, F. X., GIEDD, J. N., MARSH, W. L., HAMBURGER, S. D., SCHUBERT, A. B., VAUSS, Y. C., VAITUZIS, A. C., DICKSTEIN, D. P., SARFATTI, S. E., & RAPOPORT, J. L. (1997). Implication of Right Frontostriatal Circuitry in Response Inhibition and Attention-Deficit/Hyperactivity Disorder. *J Am Acad Child Adolesc Psychiatry*, 36(3), 374–383.
- CASEY, B. J., COHEN, J. D., JEZZARD, P., TURNER, R., NOLL, D. C., TRAINOR, R. J., GIEDD, J., KAYSEN, D., HERTZ-PANNIER, L., & RAPOPORT, J. L. (1995). Activation of Prefrontal Cortex in Children During a Nonspatial Working Memory Task with Functional MRI. *Neuroimage*, 2(3), 221–229.

- CAUFFMAN, E., & STEINBERG, L. (2000). (Im)maturity of judgment in adolescence: why adolescents may be less culpable than adults. *Behav Sci Law*, 18(6), 741–760.
- CHAMBERS, R. A., TAYLOR, J. R., & POTENZA, M. N. (2003). Developmental neurocircuitry of motivation in adolescence: a critical period of addiction vulnerability. *Am J Psychiatry*, 160(6), 1041–1052.
- CHUGANI, H.T., (1998). Biological basis of emotions: brain systems and brain development. *Pediatrics*, 102(5 Suppl E), 1225–1229.
- CONEL, J. L. (1939). The postnatal development of the human cerebral cortex (Vol. 1–6). Cambridge, MA: Harvard University Press.
- CROSBY, E. C., HUMPHREY, T., & LAUER, E. W. (1962). *Correlative anatomy of the nervous system*. New York: Macmillan.
- DAFFNER, K. R., MESULAM, M. M., SCINTO, L. F., ACAR, D., CALVO, V., FAUST, R., CHABRERIE, A., KENNEDY, B., & HOLCOMB, P. (2000). The central role of the prefrontal cortex in directing attention to novel events. *Brain*, 123 (Pt 5), 927–939.
- DIAMOND, A. (1991). Neuropsychological insights into the meaning of object concept development. In S. Carey & R. Gelman (Eds), *The epigenesis of mind: essays on biology and cognition*. Hillsdale, N.J.: L. Erlbaum Associates, 67–110.
- DIAMOND, A. (1996). Evidence for the importance of dopamine for prefrontal cortex functions early in life. *Philos Trans R Soc Lond B Biol Sci*, 351(1346), 1483–1493; discussion 1494.
- DIAMOND, A., & GOLDMAN-RAKIC, P. S. (1989). Comparison of human infants and rhesus monkeys on Piaget's AB task: evidence for dependence on dorsolateral prefrontal cortex. *Exp Brain Res*, 74(1), 24–40.
- FLECHSIG, P. (1901). Developmental (myelogenetic) localisation of the cerebral cortex in the human subject. *Lancet*, 2, 1027–1029.
- FREEMAN, W., & WATTS, J. W. (1948). The thalamic projection to the frontal lobe. *Res Publ Assoc Res Nerv, Ment Dis*, 27, 200–209.
- FUSTER, J. M. (1997). *The prefrontal cortex: anatomy, physiology and neuropsychology of the frontal lobe* (3 ed.). Philadelphia, PA: Lippincott-Raven.
- GAILLARD, W. D., HERTZ-PANNIER, L., MOTT, S. H., BARNETT, A. S., LEBIHAN, D., & THEODORE, W. H. (2000). Functional anatomy of cognitive development: fMRI of verbal fluency in children and adults. *Neurology*, 54(1), 180–185.
- GIEDD, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. *Ann N Y Acad Sci*, 1021, 77–85.
- GIEDD, J. N., BLUMENTHAL, J., JEFFRIES, N. O., RAJAPAKSE, J. C., VAITUZIS, A. C., LIU, H., BERRY, Y. C., TOBIN, M., NELSON, J., & CASTELLANOS, F. X. (1999). Development of the human corpus callosum during childhood and adolescence: a longitudinal MRI study. *Prog Neuro-psychopharmacol Biol Psychiatry*, 23(4), 571–588.

- GIEDD, J. N., RUMSEY, J. M., CASTELLANOS, F. X., RAJAPAKSE, J. C., KAYSEN, D., VAITUZIS, A. C., VAUSS, Y. C., HAMBURGER, S. D., & RAPOPORT, J. L. (1996). A quantitative MRI study of the corpus callosum in children and adolescents. *Brain Res Dev Brain Res*, 91(2), 274–280.
- GOLDBERG, E. (2001). *The executive brain: frontal lobes and the civilized mind*. Oxford: Oxford University Press.
- GOLDMAN-RAKIC, P. S. (1981). Prenatal formation of cortical input and development of cytoarchitectonic compartments in the neostriatum of the rhesus monkey. *J Neurosci*, 1(7), 721–735.
- GOLDMAN-RAKIC, P. S., & BROWN, R. M. (1981). Regional changes of monoamines in cerebral cortex and subcortical structures of aging rhesus monkeys. *Neuroscience*, 6(2), 177–187.
- GOLDMAN-RAKIC, P. S., & PORRINO, L. J. (1985). The primate mediodorsal (MD) nucleus and its projection to the frontal lobe. *J Comp Neurol*, 242(4), 535–560.
- GOMEZ-PEREZ, E., OSTROSKY-SOLIS, F., & PROSPERO-GARCIA, O. (2003). The development of attention, memory and the inhibitory processes: the chronological relation with the maturation of brain structure and functioning. *Revista de Neurologia*, 37(6), 561–567.
- GRIFFIN, P. (2003). *Trying and Sentencing Juveniles as Adults: An Analysis of State Transfer and Blended Sentencing Laws*. Pittsburgh, PA: National Center for Juvenile Justice.
- GRUBER, S. A., ROGOWSKA, J., HOLCOMB, P., SORACI, S., & YURGELUN-TODD, D. (2002). Stroop Performance in Normal Control Subjects: an fMRI Study. *NeuroImage*, 16(2), 349–360.
- GRUBER, S. A., ROGOWSKA, J., & YURGELUN-TODD, D. A. (2004). Decreased activation of the anterior cingulate in bipolar patients: an fMRI study. *J Affect Disord*, 82(2), 191–201.
- GUR, R. C., GUNNING-DIXON, F. M., TURETSKY, B. I., BILKER, W. B., & GUR, R. E. (2002). Brain region and sex differences in age association with brain volume: a quantitative MRI study of healthy young adults. *Am J Geriatr Psychiatry*, 10(1), 72–80.
- HALPERN, J., & FINGER, W. R. (1992). Prevention of STDs—the challenge of changing behaviors. *Network*, 12(4), 16–18.
- HARIRI, A. R., BOOKHEIMER, S. Y., & MAZZIOTTA, J. C. (2000). Modulating emotional responses: effects of a neocortical network on the limbic system. *NeuroReport*, 11(1), 43–48.
- HUTTENLOCHER, J., HEDGES, L. V., & PROHASKA, V. (1992). Memory for day of the week: a 5 + 2 day cycle. *J Exp Psychol Gen*, 121(3), 313–325.
- HUTTENLOCHER, P., & DABHOLKAR, A. (1997). Developmental anatomy of prefrontal cortex. In N. Krasnegor, G. R. Lyon & P. S. Goldman-Rakic (Eds), *Development of the prefrontal cortex*. Baltimore: Paul H. Brookes Publishing Co., 69–83.

- JACOBSEN, C.F. (1935). Functions of frontal associative area in primates. *Arch of Neur Psych*, 33, 558–569.
- JERNIGAN, T. L., TRAUNER, D. A., HESSELINK, J. R., & TALLAL, P. A. (1991). Maturation of human cerebrum observed in vivo during adolescence. *Brain*, 114 (5), 2037–2049.
- KILLGORE, W. D., OKI, M., & YURGELUN-TODD, D. A. (2001). Sex-specific developmental changes in amygdala responses to affective faces. *Neuroreport*, 12(2), 427–433.
- KILLGORE, W. D., & YURGELUN-TODD, D. (2005). Developmental changes in the functional brain responses of adolescents to images of high and low calorie foods. *Dev Psychobiol*, 47(4), 377–397.
- KLINGBERG, T., VAIDYA, C. J., GABRIELI, J. D. E., MOSELEY, M. E., & HEDEHUS, M. (1999). Myelination and organization of the frontal white matter in children: a diffusion tensor MRI study. *NeuroReport*, 10(13), 2817–2821.
- KUYPERS, H. G., SZWARCBART, M. K., MISHKIN, M., & ROSVOLD, H. E. (1965). Occipitotemporal Corticocortical Connections In The Rhesus Monkey. *Exp Neurol*, 11, 245–262.
- LARROCHE, J. C., & AMIEL, C. (1966). Thrombosis of the sylvian artery during the neonatal period. Anatomical study and pathogenic discussion of so-called congenital hemiplegia. *Arch Fr Pediatr*, 23(3), 257–274.
- LEONARD, C. M. (1969). The prefrontal cortex of the rat. I. Cortical projection of the mediodorsal nucleus. II. Efferent connections. *Brain Res*, 12(2), 321–343.
- LEZAK, M. D. (2004). *Neuropsychological Assessment* (4th ed.). New York: Oxford University Press.
- LUNA, B., THULBORN, K. R., MUNOZ, D. P., MERRIAM, E. P., GARVER, K. E., MINSHEW, N. J., KESHAVAN, M. S., GENOVESE, C. R., EDDY, W. F., & SWEENEY, J. A. (2001). Maturation of Widely Distributed Brain Function Suberves Cognitive Development. *NeuroImage*, 13(5), 786–793.
- LURIA, A. R. (1966). *Higher cortical functions in man*. New York: Basic Books.
- MARIN-PADILLA, M. (1970). Prenatal and early postnatal ontogenesis of the human motor cortex: a golgi study. I. The sequential development of the cortical layers. *Brain Res*, 23(2), 167–183.
- MARIN-PADILLA, M. (1988). Early ontogenesis of the human cerebral cortex. In A. Peters & E. G. Jones (Eds), *Cerebral cortex* (Vol. 7). New York: Plenum, 1–34.
- MC, L. T. (1950). Thalamic projection to frontal cortex in man. *J Neurol Neurosurg Psychiatry*, 13(3), 198–202.
- MEYER-LINDENBERG, A. (1996). The evolution of complexity in human brain development: an EEG study. *Electroencephalogr Clin Neurophysiol*, 99(5), 405–411.

- MILLER, E. K. (2000). The Prefrontal Cortex and Cognitive Control. *Nat Rev Neurosci*, 1(1), 59–65.
- MRZLJAK, L., UYLINGS, H. B., VAN EDEN, C. G., & JUDAS, M. (1990). Neuronal development in human prefrontal cortex in prenatal and postnatal stages. *Prog Brain Res*, 85, 185–222.
- NELSON, C. A., MONK, C. S., LIN, J., CARVER, L. J., THOMAS, K. M., & TRUWIT, C. L. (2000). Functional Neuroanatomy of Spatial Working Memory in Children. *Dev Psychol*, 36(1), 109–116.
- PFEFFERBAUM, A., MATHALON, D. H., SULLIVAN, E. V., RAWLES, J. M., ZIPURSKY, R. B., & LIM, K. O. (1994). A Quantitative Magnetic Resonance Imaging Study of Changes in Brain Morphology From Infancy to Late Adulthood. *Arch Neurol*, 51(9), 874–887.
- PIAGET, J. (1954). The development of time concepts in the child. *Proc Annu Meet Am Psychopathol Assoc*, 34–44; discussion, 45–55.
- PRIBAM, K. H., CHOW, K. L., & SEMMES, J. (1953). Limit and organization of the cortical projection from the medial thalamic nucleus in monkey. *J Comp Neurol*, 98, 433–448.
- RUBIA, K., OVERMEYER, S., TAYLOR, E., BRAMMER, M., WILLIAMS, S. C., SIMMONS, A., ANDREW, C., & BULLMORE, E. T. (2000). Functional frontalisation with age: mapping neurodevelopmental trajectories with fMRI. *Neurosci Biobehav Rev*, 24(1), 13–19.
- SCHADE, J. P., & VAN, G. W. (1961). Structural organization of the human cerebral cortex. 1. Maturation of the middle frontal gyrus. *Acta Anat (Basel)*, 47, 74–111.
- SCHETKY, D. H., & BENEDEK, E. P. (2002). *Principles and Practice of Child and Adolescent Forensic Psychiatry*. Washington, D.C.: American Psychiatric Pub.
- SCHLAGGAR, B. L., BROWN, T. T., LUGAR, H. M., VISSCHER, K. M., MIEZIN, F. M., & PETERSEN, S. E. (2002). Functional Neuroanatomical Differences Between Adults and School-Age Children in the Processing of Single Words. *Science*, 296(5572), 1476–1479.
- SCOTT, E.S., & GRISSO, T. (1997). The Evolution of Adolescence: A Developmental Perspective on Juvenile Justice Reform. *J. Crim. L. & Criminology*, 88, 137–189.
- SEAGRAVE, D., & GRISSO, T. (2002). Adolescent development and the measurement of juvenile psychopathy. *Law Hum Behav*, 26(2), 219–239.
- SIDMAN, R. L., & RAKIC, P. (1973). Neuronal migration, with special reference to developing human brain: a review. *Brain Res*, 62(1), 1–35.
- SOWELL, E. R., DELIS, D., STILES, J., & JERNIGAN, T. L. (2001). Improved memory functioning and frontal lobe maturation between childhood and adolescence: a structural MRI study. *J Int Neuropsychol Soc*, 7(3), 312–322.

- SOWELL, E. R., THOMPSON, P. M., HOLMES, C. J., JERNIGAN, T. L., & TOGA, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nat Neurosci*, 2(10), 859–861.
- SOWELL, E. R., THOMPSON, P. M., TESSNER, K. D., & TOGA, A. W. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: Inverse relationships during postadolescent brain maturation. *J Neurosci*, 21(22), 8819–8829.
- STEINBERG, L., & SCOTT, E. S. (2003). Less guilty by reason of adolescence: developmental immaturity, diminished responsibility, and the juvenile death penalty. *Am Psychol*, 58(12), 1009–1018.
- THOMAS, K. M., KING, S. W., FRANZEN, P. L., WELSH, T. F., BERKOWITZ, A. L., NOLL, D. C., BIRMAHER, V., & CASEY, B. J. (1999). A Developmental Functional MRI Study of Spatial Working Memory. *NeuroImage*, 10(3 Pt 1), 327–338.
- UYLINGS, H. B., & VAN EDEN, C. G. (1990). Qualitative and quantitative comparison of the prefrontal cortex in rat and in primates, including humans. In H. B. Uylings, C. G. van Eden, J. P. C. De Bruin, M. A. Corner & M. G. P. Feenstra (Eds), *The prefrontal cortex: its structure, function and pathology*. Amsterdam: Elsevier, 31–62.
- VAN BUREN, J. M., & BORKE, R. C. (1972). Variation and connection of the human thalamus. Berlin: Springer.
- VAN DER STELT, O., KOK, A., SMULDERS, F. T., SNEL, J., & BOUDEWIJN GUNNING, W. (1998). Cerebral event-related potentials associated with selective attention to color: Developmental changes from childhood to adulthood. *Psychophysiology*, 35(3), 227–239.
- VIRGINIA DEPARTMENT OF HEALTH—OFFICE OF FAMILY HEALTH SERVICES. (2005). Adolescent Health Programs of the Virginia Department of Health. At <http://www.vahealth.org/adolescenthealth/>. (Last accessed October 13, 2005).
- WALKER, A. E. (1938). *The primate thalamus*. Chicago: University of Chicago Press.
- WAXMAN, S. G., & FOSTER, R. E. (1980). Development of the axon membrane during differentiation of myelinated fibers in spinal nerve roots. *Proc R Soc Lond B Biol Sci*, 209(1176), 441–446.
- YAKOVLEV, P. I., & LECOURS, A. R. (1967). The myelogenetic cycles of regional maturation of the brain. In A. Minkowski (Ed), *Regional development of the brain in early life*. Oxford: Blackwell, 3–70.
- YURGELUN-TODD, D. A., KILLGORE, W. D., & CINTRON, C. B. (2003). Cognitive correlates of medial temporal lobe development across adolescence: a magnetic resonance imaging study. *Percept Mot Skills*, 96(1), 3–17.
- YURGELUN-TODD, D. A., KILLGORE, W. D., & YOUNG, A. D. (2002). Sex differences in cerebral tissue volume and cognitive performance during adolescence. *Psychol Rep*, 91(3 Pt 1), 743–757.

- YURGELUN-TODD, D. A., & RENSHAW, P. F. (2000). Magnetic resonance spectroscopy in childhood psychiatric disorders. In M. Ernst & J. M. Rumsey (Eds), *Functional Neuroimaging in Child Psychiatry* (pp. 59–76). Cambridge, UK: Cambridge University Press, 59–76.